Please delete the paragraph at page 27, lines 24-26, and replace the following paragraph therefore:

"Anthracyclines include daunorubicin (formerly daunomycin) and doxorubicin (also referred to herein as ADRIAMYCIN (doxorubicin HCl). Additional examples include mitozantrone and bisantrene."

Please delete the paragraph at page 28, lines 8-11, and replace the following paragraph therefore:

"Further examples of cytotoxic agents include, but are not limited to, ricin, bryodin, gelonin, supporin, doxorubicin, TAXOL, cytochalasin B, gramicidin D, ethidium bromide, etoposide, tenoposide, colchicine, digydroxy antracin dione, l- dehydrotesteosterone, and glucocoticoid."

Please delete the paragraph at page 48, lines 23-29, and replace the following paragraph therefore:

"Results of the CDC demonstrate that mutant hBR96-2B has approximately 10 fold less activity than the control hBR96-1 (which has two affinity mutations, one in H2 and one in H3, as shown in provisional patent application Serial # 60/023,033 filed August 2, 1996 (Figure 20)). The mutants that have the least ability to kill cells in the presence of complement is hBR96-2C with the triple mutations at positions 318, 320 and 322 and the hBR96-2H mutant (least cytotoxic antibodies in the panel) which contains all six mutations at the three different locations. ADCC activity was most affected by the CH2 deleted hBR96-2 molecule (Figure 21). hBR96-2B and –2H lost between 100 and 1000 fold activity to kill in the presence of effector cells. In the ADCC assay the hBR96-2B molecule also lost approximately 10 fold activity (Figure 21)."

IN THE CLAIMS:

Please cancel claim 7, without prejudice.

Please rewrite the following claims:

1. (Amended) A method for inhibiting immunoglobulin-induced toxicity resulting from immunoglobulin immunotherapy in a subject comprising administering an immunoglobulin molecule to the subject, the immunoglobulin molecule having a variable region and a constant region, the immunoglobulin molecule being modified prior to administration by structurally altering multiple toxicity-associated regions in the CH2 domain so that immunoglobulin-induced toxicity is inhibited.